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The role of nutrition in the etiology of human disease, including cancer, has been recognized for a very long time. This knowledge and, perhaps, personal preference are reflected by the choice and balance of edibles included in the daily diet.

Considerable progress has been made in identifying the cellular targets involved in interaction between cells and initiating carcinogens and promoters. It has been evident since the work of Miller and Miller (1969) that modification of cellular DNA (leading to mutagenesis) is an important event in initiating carcinogenesis. There are, of course, exceptions to the correlation between mutagenicity and carcinogenicity. Some chemicals show strong mutagenicity when tested in in vitro test systems, but they do not induce cancer in animals.

Several chapters of this volume are devoted to the understanding of the mechanisms of carcinogenicity and anticarcinogenicity, as well as the role of diet or hormones in the induction or inhibition of carcinogenesis.

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MECHANISMS OF CARCINOGENICITY AND ANTICARCINOGENICITY:

ROLE OF DIETARY COMPONENTS

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The role of nutrition in the etiology of human disease, including cancer, has been recognized for a very long time. This knowledge and, perhaps, personal preference are reflected by the choice and balance of edibles included in the daily diet. Even at present, in certain societies where strong emphasis on traditional values has preserved the dietary patterns of ancient times, the daily diet of most people includes a wide variety of legumes, high in protease inhibitors, and selected vegetables, high in β-carotene. With our current state of knowledge, we can appreciate the role of tobacco in causing lung cancer and the role of betel quid in the etiology of oral cancer. It may be of interest to note that in certain societies, the use of tobacco and betel quid was strictly prohibited as early as the sixteenth-century because of the suspicion that these agents may be involved in the etiology of cardiovascular diseases and cancer.

Recent epidemiological studies conducted under diverse cultural conditions have shown marked differences in geographic distribution of specific types of cancer. The higher incidences of cancer have further been associated with higher consumption of certain categories of food and/or their method of preparation (1,3). The difference between the cancer incidence among immigrants with their traditional dietary habits and that of the local population has given further credence to the role of diet in causing cancer.

Our scientific views on the role of nutrition in cancer induction or prevention are based on the studies undertaken by Tannenbaum and his colleagues in the early 1940s. Although their studies were concerned with caloric intake and cancer, today we know that a variety of nutritional components are involved in the initiation or intervention, progression, or regression of cancer. The protective role of certain dietary factors such as vitamin E, carotene, selenium, glutathione, ascorbic acid, uric acid, phenols, and tocopherols is noteworthy in this regard.

Considerable progress has been made in identifying the cellular targets involved in interaction between cells and initiating carcinogens and promotors. It has been evident since the work of Miller and Miller (2) that modification of cellular DNA (leading to mutagenesis) is an important event in initiating carcinogenesis. There are, of course, exceptions to the correlation between mutagenicity and carcinogenicity. Some chemicals show strong mutagenicity when tested in in vitro test systems, but they do not induce cancer in animals. Similarly, certain carcinogens do not cause gene mutation, but induce their effects either through induction of chromosome aberrations, including aneuploidy, or alteration of processes leading to the synthesis or function of DNA, or through other less direct mechanisms.

We are becoming increasingly aware that carcinogenesis is an extremely complex phenomenon mediated through genetic and epigenetic mechanisms. Cancer is an outcome of an interaction between a specific genotype and a specific chemical (or physical) agent. The problem of mechanistic understanding of chemical carcinogenesis becomes even more intricate when we consider the fact that man is seldom exposed to individual chemicals but always to an array of chemical mixtures. As a result the biological effects of complex chemical mixtures are difficult to comprehend and predict.

Several chapters of this volume are devoted to the understanding of the mechanisms of carcinogenicity and anticarcinogenicity, as well as the role of diet or hormones in the induction or inhibition of carcinogenesis.

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